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## BLOOD LIPID PROFILE AS A DIAGNOSTIC MARKER OF ACUTE PANCREATITIS IN DOGS

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**Abstract.** Acute pancreatitis is a common non-communicable pathology in dogs, the untimely diagnosis and treatment of which is lethal (27-58% of cases). One of the most common medical tests for acute pancreatitis is to determine the concentration of triglycerides and other fractions of blood lipids. The purpose of the study was to investigate changes in lipid fractions and other biochemical parameters in dogs with acute pancreatitis. For the experiment, two groups of dogs of 10 individuals each were formed. The control group included healthy animals, the experimental group – dogs with acute pancreatitis. It was found that in dogs with acute pancreatitis, the concentration of triglycerides increased by 67% compared with healthy individuals. Cholesterol and phospholipid concentrations also increased by 23 and 9%, respectively. Inflammatory processes in the pancreas led to the development of cytolytic processes in its acini, as well as hepatocytes of the liver, as indicated by increased activity of indicator enzymes ALT, AST, LF and bilirubin. In addition, in sick dogs, the protein-synthesising function of the liver was impaired: a reduced proportion of albumin was registered against the background of increased total serum protein. Acute pancreatitis leads to a decrease in insulin synthesis, which is manifested by hyperglycaemia – in sick dogs, glucose levels are probably twice as high as in healthy animals. In pancreatitis, the violation of digestion and absorption of nutrients caused by intensive use of high-energy compounds (in particular, creatine phosphate) and an increase in creatinine (3.8 times). At the same time, the catabolism of proteins increased and the products of their intermediate and final metabolism – ammonia and urea – accumulated (by 60 and 57%, respectively), which is a sign of reduced filtration capacity of the kidneys. In addition, in acute pancreatitis within the physiological norm, the concentration of calcium decreased, and inorganic phosphorus – increased

**Keywords:** triglycerides, cholesterol, phospholipids, indicator enzymes, pancreas



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## INTRODUCTION

Pancreatitis is a fairly common pathology in dogs. Factors underlying the pathogenesis of pancreatitis are diverse: stress, eating disorders, inflammatory processes of the gastrointestinal tract, liver disease, etc. [1]. This disease can be a secondary complication of infectious and parasitic pathologies, especially those that have a strong development and involve in the general pathogenesis of the systemic inflammatory response of the organism [2]. In particular, pancreatitis is often accompanied by babesiosis, heartworm disease, distemper, and viral enteroinfections in dogs, which are typical pathologies of infectious origin in the Polissya region of Ukraine [3; 4]. There are two forms of pancreatitis: acute and chronic. Acute form is characterised by a high percentage of deaths – 27-58% [5], chronic – significantly worsens the quality of life of animals and creates inconvenience for their owners.

The pathogenesis of pancreatitis is not fully understood. Most theories explain the apoptosis of pancreatic cells by oxidative stress, the action of products of nitrogen metabolism – ammonia and creatinine, impaired blood supply to the gland [6]. There is a hypothesis that links the occurrence of pancreatitis with calcium deficiency in the blood caused by the synthesis of its salts with fatty acids. As a result of negative reverse regulation, the secretion of parathyroid hormone increases, which causes an increase in the content of calcium in the cells of the gland and leads to their further destruction [7]. Due to the cytolysis of glandular cells of the pancreas, the activity of enzymes increases in the blood, but decreases in the intestine. Affected dogs have impaired digestion and absorption of nutrients and develop characteristic symptoms such as loss of appetite, vomiting, flatulence, diarrhoea, or constipation [8].

Pancreatitis also causes destructive changes in other organs, including the liver, kidneys, lungs, which significantly complicates the diagnosis of this pathology. For example, any liver dysfunction also leads to an increase in cytolytic phenomena and the release of enzymes into the blood, disorders of the inclusion of triglycerides and cholesterol in lipoproteins [9]. Acute pancreatitis develops quite rapidly, so in the case of incorrect diagnosis and therapy, irreversible changes may appear in the animal body. However, the diagnosis of pancreatitis, even in the acute form, is associated with certain difficulties, as there are no specific markers of the disease. Thus, the main marker of pancreatitis in humans and some animals is the activity of pancreatic  $\alpha$ -amylase, but in dogs its activity in the blood does not reflect the degree of pathological process in the pancreas [10; 11].

At the present stage, one of the most effective criteria for diagnosing pancreatitis in dogs is to determine the activity of Specific Canine Pancreatic Lipase Immunoreactivity (Spec cPL or cPLI) [12; 5]. The

sensitivity of this method is 80-85%. However, the activity of the enzyme may increase in other pathological conditions – duodenitis, the presence of a foreign body in the small intestine, its mechanical damage – all these factors disrupt the integrity of the intestine and provoke the release of pancreatic lipase into the blood. In addition, the definition of Spec cPL requires equipment for immunochemical testing and is not always available to veterinary laboratories. The transfer of pancreatic lipase into the blood causes a decrease in its excretion into the intestinal cavity, resulting in reduced breakdown and absorption of triglycerides.

The concentration of triglycerides and cholesterol is one of the markers of acute pancreatitis [13; 14]. Inflammatory processes in the pancreas are characterised by an increase in the concentration of proteins of the acute phase of inflammation, in particular C-reactive protein [15-17].

*The purpose of the study* was to investigate the content of lipid fractions and other serum biochemical parameters as a complex of biochemical markers for acute pancreatitis in dogs.

*The objective of the study* was to determine the concentration of triglycerides, cholesterol, phospholipids, total protein, glucose, bilirubin, activity of indicator enzymes ALT (alanine aminotransferase), AST (asparagine aminotransferase), LF (alkaline phosphatase, blood phosphatase, with those in healthy animals).

## MATERIALS AND METHODS

The studies were conducted during 2018-2020 based on the research and production veterinary clinic and the Department of Parasitology, Veterinary and Sanitary Expertise, Zoohygiene of Polissya National University. For experiments, two groups of dogs, 10 heads each, were formed. The groups included 5 females and 5 males, each group had both sterilised and non-sterilised individuals. The age of the animals ranged from 3 to 8 years. The animals were of different breeds and crossbreeds.

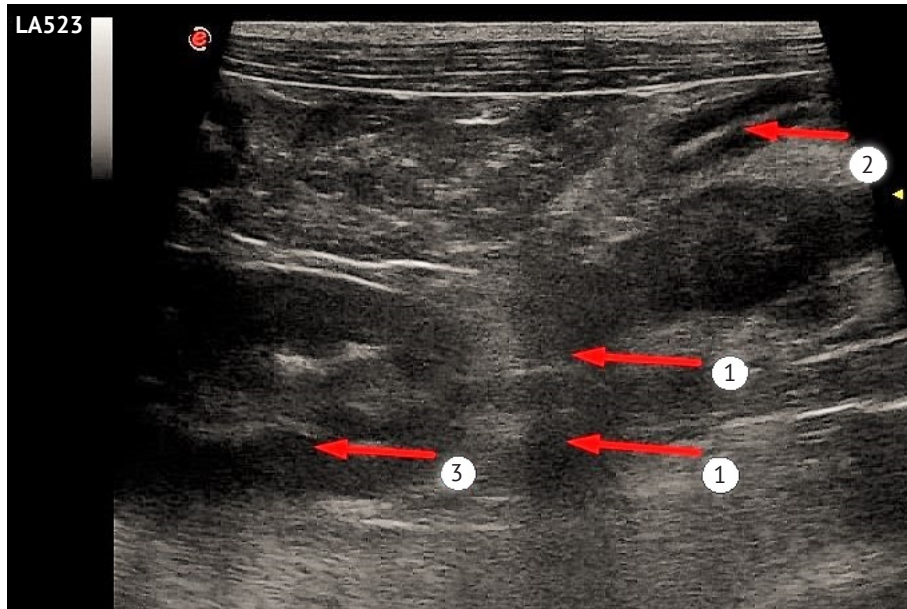
The control group included clinically healthy dogs that underwent a preventive examination in the clinic. Experimental group included dogs diagnosed with acute pancreatitis according to history, clinical examination, special and laboratory studies.

The clinical study was performed according to general methods: examination, thermometry, palpation, percussion, auscultation. In the experimental group, the characteristic clinical signs of pancreatitis were: deterioration or loss of appetite, signs of nausea, vomiting, diarrhoea or constipation, sometimes fever. Pain was determined in the abdominal cavity of sick dogs. Such signs are not specific exclusively to pancreatitis, and specific and laboratory methods of animal study were used to further clarify the diagnosis.

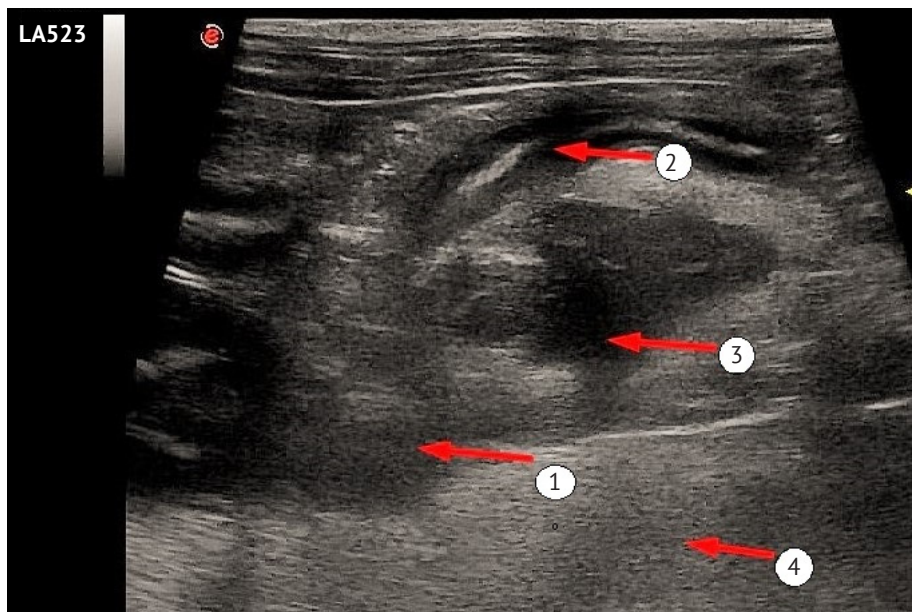
Special methods included ultrasound using the SonoScape S11 ultrasound diagnostic scanner with

Doppler system (SonoScape Medical Corp., Russian Federation). Animal testing was carried out in the morning, on an empty stomach, when there is the least amount of unnecessary factors that interfere with the detection of the pancreas. The disappearance of the contours of the abdominal cavity in the upper right and

the presence of a “signal loop” are diagnostic markers of pancreatitis. Determining criteria for the presence of pancreatitis were hypoechoic parenchyma of the pancreas, hyperechogenic peripancreatic adipose tissue, bile duct obstruction (Fig. 1-2) [18].



**Figure 1.** Sonogram of the right dorsal abdomen of the dog with acute pancreatitis: 1 – blurred borders and hypoechoic structure of the pancreas, 2 – splenic vein, 3 – duodenal area filled with gases (“signal loop”)



**Figure 2.** Sonogram of the pancreas of the dog with acute pancreatitis: 1 – erased boundaries of the right lobe of the pancreas, 2 – splenic vein, 3 – aorta, 4 – peripancreatic hyperechogenic adipose tissue

Blood from animals of both experimental groups was collected from the subcutaneous forearm vein (vena subcutanea antebrachii) in accordance with the rules of asepsis and antiseptics. Disposable puncture needles

and Vacusera vacuum tubes with lithium heparin and clean ones were used. For experimental studies, blood serum was used to determine the concentration of total protein, albumin, glucose, total bilirubin, creatinine,

cholesterol, the activity of the enzymes alanine aminotransferase (ALT), aspartate aminotransferase (AST) (Reitman-Frenkel method), alkaline phosphatase (LP) (kinetic method), calcium (by the colorimetric method of a colour compound in the reaction of calcium with o-cresolphthalein in an alkaline medium), inorganic phosphorus (Briggs colorimetric method). The studies were carried out using a semi-automatic biochemical analyser Chem 7 (Erba, Czech Republic) with a line of reagents from DAC (Republic of Moldova).

The concentration of triglycerides was determined by enzymatic colorimetric method (PAP) using a set of reagents "Triglycerides PAP" manufactured by "Diagnosticum" company (Hungary).

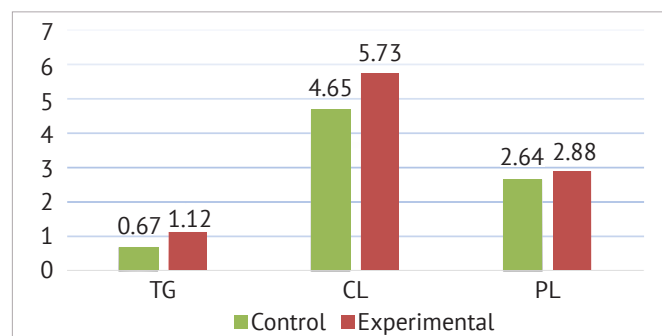
The concentration of phospholipids was determined by extraction of the lipid fraction with isoamyl alcohol, followed by mineralisation. Inorganic phosphorus in interaction with ammonium molybdate

forms phosphomolybdic acid, the optical density of which was measured at wavelengths  $\lambda = 630$  nm.

The obtained results were statistically processed using the Statistica 13.3 software package. Data were checked for normal distribution using the Distribution Fitting module. The arithmetic mean value, its standard error were determined. Estimation of regression equality was performed according to Fisher's method (ANOVA) for a significance level of 0.05.

## RESULTS AND DISCUSSION

Destruction of pancreatic cells in acute pancreatitis is accompanied by the excretion of some enzymes into the blood (instead of the small intestine), while some nutrients remain undigested and partly excreted in the faeces, and partly enter the blood [1]. The severity of acute pancreatitis correlates with the concentration of various lipid fractions, in particular serum triglycerides (Fig. 3).



**Figure 3.** The content of triglycerides (TG), cholesterol (CL), and phospholipids (PL) in the serum of dogs with acute pancreatitis, mmol/l

The concentration of all studied fractions of serum lipids in dogs of the experimental group tended to increase compared to those in the control. The largest difference between the groups was observed in the concentration of triglycerides ( $p < 0.05$ ). Thus, in animals with pancreatitis, the concentration of this fraction was almost twice that of healthy animals. The smallest changes were found in the concentration of phospholipids – 9%. Notably, in different animals, especially in the experimental group, there was a high degree of variance, which indicates the individual mechanisms of regulation of lipid metabolism in dogs with this pathology.

Pancreatitis is a pathology that affects most parts of the metabolism and the functional capacity of many organs. This primarily applies to the liver. To assess the condition of the liver, markers of its functional activity were determined (Table 1). The concentration of total protein in the dogs of the experimental group was higher ( $P \geq 0.05$ ) than in the control group, but was within the reference range. At the same time, the concentration of albumin in sick dogs significantly decreased. Glucose concentration in the control group was within physiological values. In animals of the experimental group, its content was twice the control index ( $P \leq 0.05$ ).

**Table 1.** Biochemical markers of liver function in healthy dogs and with acute pancreatitis

Indicator	Healthy dogs (n=10)	Dogs with acute pancreatitis (n=10)
Total protein, g/l	62.4±7.27	69.8±6.42
Albumins, g/l	32.6±4.27	22.8±3.91*
Glucose, mmol/l	5.8±1.29	11.7±2.37*
Total bilirubin, $\mu$ mol/l	7.2±0.93	29.5±3.15**
ALAT, u/l	31.6±3.41	72.7±6.84*
AST, u/l	29.7±12.60	74.9±19.81*
LF, u/l	71.5±22.74	101.4±25.38

**Note:** \*  $P \leq 0.05$ , \*\*  $P \leq 0.01$  for healthy dogs

The content of total bilirubin in the serum of experimental animals significantly exceeded physiological values and was 4.1 times higher than in control group. The activity of both transaminases (ALT and AST) in sick dogs significantly increased compared to the control group, exceeding the upper limit of physiological values. LF activity had a similar trend, but its growth in the dogs of the experimental group was not as rapid as

the activity of transaminases and was 41.8% compared with control group.

The excretory system is another system of organs in which the intensity of metabolic processes during pancreatitis has increased. To assess the functional capacity of this system, the content of creatinine, urea, and ammonia was studied (Table 2).

**Table 2.** Some biochemical markers of the functional state of the kidneys in healthy dogs and with acute pancreatitis

Indicator	Healthy dogs (n=10)	Dogs with acute pancreatitis (n=10)
Creatinine, $\mu\text{mol/l}$	63.5 $\pm$ 10.31	238.5 $\pm$ 38.76**
Urea, mmol/l	6.9 $\pm$ 1.91	22.5 $\pm$ 4.52*
Ammonia, $\mu\text{mol/l}$	27.3 $\pm$ 3.51	42.8 $\pm$ 6.33*

**Note:** \*  $P \leq 0.05$ , \*\*  $P \leq 0.01$  for healthy dogs

The concentration of creatinine in the serum of dogs in the experimental group was 3.8 times higher than in the control group ( $P \leq 0.01$ ). Furthermore, the content of ammonia (one of the end products of protein metabolism) in the blood of experimental dogs was 1.6 times higher ( $P \leq 0.05$ ) than in control animals. The

content of urea, which is synthesised by the liver from ammonia, was also higher in sick dogs – an increase of 1.57 times ( $P \leq 0.05$ ). Calcium ions and phosphates play an important role in the pathogenesis of acute pancreatitis (Table 3).

**Table 3.** The content of calcium and inorganic phosphorus in the serum of healthy dogs and with acute pancreatitis

Indicator	Healthy dogs (n=10)	Dogs with acute pancreatitis (n=10)
Calcium, mmol/l	2.74 $\pm$ 0.38	2.18 $\pm$ 0.51
Inorganic phosphorus, mmol/l	1.53 $\pm$ 0.29	2.05 $\pm$ 0.62

Calcium content in sick animals was lower than in healthy ones ( $P \geq 0.05$ ) and was at the lower limit of physiological values. Inorganic phosphorus, on the other hand, was higher in dogs with acute pancreatitis than in healthy ones ( $P \geq 0.05$ ), but did not exceed the reference values.

Serum lipids are considered one of the informative markers in the diagnosis of acute pancreatitis in dogs [19]. Inflammatory processes in the pancreas lead to a decrease in the synthesis and excretion of enzymes that catalyse the hydrolysis of lipids of different fractions: triglycerides, cholesterol ethers and phospholipids. As a result, their inclusion in lipoproteins (the main transport form of lipids in the blood) is disrupted, so their content in the blood increases [2]. It is believed that a deficiency of exogenous lipids causes the release of endogenous lipids from the fat depot [13]. According to some studies, the degree of inflammation correlates with an increase in the content of all fractions of lipids, especially triglycerides [20]. Own results showed that in dogs with acute pancreatitis, the concentration of all studied lipid fractions increased. The maximum degree of growth (1.67 times) was demonstrated by triglycerides (fats), which are the main fraction of dietary lipids.

An analysis of the scientific literature has shown

that there is no single mechanism to explain such changes [11]. According to some authors, the increase in triglycerides is conditioned by a decrease in the synthesis of the enzyme lipase, which hydrolyses fats, and also by cytolysis of secretory cells of the pancreas, resulting in a significant part of lipase entering the blood [21; 13].

The increase in the concentration of other fractions – cholesterol and phosphatides in the blood of sick dogs was slightly lower, respectively – 1.23 and 1.09 times. Such changes may be due to the fact that the enzymes phospholipase, cholesterol are more synthesised by the mucous membrane of the small intestine [14]. Impaired absorption of cholesterol (basic metabolite, source of bile acid synthesis) reduces the intensity of emulsification [14]. Decreased absorption of phospholipids disrupts the development of chylomicrons and lipoproteins. This, in turn destabilises the transport of triglycerides through the blood to the fat depot [21].

Acute pancreatitis has a destructive effect on the cells of the islet tissue, resulting in reduced synthesis of pancreatic hormones [1]. Decreased insulin levels lead to hyperglycaemia. Thus, in dogs with pancreatitis, the concentration of glucose in the blood serum twice exceeded that in healthy animals. Hyperglycaemia has

another side – it reduces the amount of glucose that comes from the blood to tissues, including the liver. At the same time, it should be noted that glucose plays an important role in the detoxification functions of the liver [13]. In addition, pancreatitis increases the build-up of toxic products of incomplete digestion of nutrients. Impaired absorption of nutrients before their entry into the colon leads to the putrefactive processes, resulting in the development of toxic products of amino acid metabolism [8].

In the experiment, the increase in the intensity of protein catabolism led to the accumulation of intermediate and final products of nitrogen metabolism. In dogs with acute pancreatitis, the concentration of creatinine, urea, and ammonia increased. The concentration of creatinine increased the most rapidly – 3.8 times. Such changes are caused by an energy deficit in the muscles and, consequently, an increase in the use of creatine phosphate. The accumulation of creatinine in the blood of sick dogs indicates impaired renal function, in particular a decrease in their filtration capacity [5].

Urea concentration is a marker of the condition of both the kidneys and the liver. Urea is synthesised in the liver, but excreted by the kidneys in the urine. Increased urea in animals with acute pancreatitis, as in the case of creatinine, indicates renal congestion. In addition, an increase in the concentration of ammonia in the blood may be an indicator of the inability of the liver to fully utilise this toxic metabolite [22; 23]. This hypothesis about the stress of metabolic processes in the liver is confirmed by other indicators. Although the concentration of total protein in sick animals increased due to dehydration, the absolute content of albumin decreased, which is a sign of impaired liver protein synthesis. At the same time, an increase in the serum activity of the indicator enzymes ALT, AST and LF, which are markers of the cytolytic syndrome, was detected [15].

A study of the concentration of total bilirubin showed that in dogs with acute pancreatitis it is more than four times higher than in healthy dogs. It is an evidence of destructive phenomena in the liver parenchyma and bilirubin in the blood. Thus, acute pancreatitis causes liver and kidney dysfunction.

Calcium is one of the factors involved in the pathogenesis of acute pancreatitis [24]. Some of the calcium ions in the intestinal cavity form insoluble compounds with fatty acids and the concentration of this element in the blood decreases. In this study, the concentration in the blood of sick animals was 20% lower than in healthy, which coincides with the findings of similar studies [24]. Hypocalcemia reflexively causes

an increase in calcium excretion not only in plasma but also in cell compartments, in particular the pancreatic parenchyma [24]. The accumulation of Calcium in the cytoplasm of cells leads to the release of potassium ions into the intercellular space, disruption of membrane potential, metabolism, and cell death [6]. Decreased serum calcium and increased in cells is one of the factors in the further development of pancreatitis [25].

Along with the decrease in calcium content, there was an increase (within physiological limits) in the concentration of inorganic phosphorus in the blood of dogs with acute pancreatitis (+34% compared to healthy animals). The increase in inorganic blood phosphates is the result of the decomposition of phosphorous compounds, in particular high-energy (creatine phosphate and others), depletion of energy reserves for pancreatitis [25]. Notably, the real increase in the content of inorganic phosphates may be greater, because parathyroid hormone stimulates the excretion of phosphates in the urine and thus the visible content does not reflect the degree of its development.

## CONCLUSIONS

1. In dogs with acute pancreatitis the concentration of the following lipid fractions increases in blood: the maximum growth is characteristic of triglycerides (+67%), cholesterol (+23%), and the smallest growth – for phospholipids (+9%). An increase in the concentration of triglycerides in the blood can be used as a diagnostic marker of acute pancreatitis.

2. The combination of metabolic changes caused by the development of acute pancreatitis leads to impaired renal function and accumulation of nitrogen metabolism products: creatinine – 3.8 times, ammonia (+60%), urea (+57%), which indicates a decrease in filtration capacity of the kidneys.

3. In acute pancreatitis in dogs, violations of the functional ability of the liver occur, the concentration of albumin decreases, the activity of indicator enzymes (ALT, AST, LF), bilirubin increases. This is a sign of the development of cytolytic syndrome in liver cells due to the accumulation of metabolic products of nitrogen-containing compounds.

4. The pathogenesis of acute pancreatitis leads to changes in calcium metabolism, resulting in a decrease in its concentration in the blood. The concentration of inorganic phosphorus, on the contrary, increased significantly, which is the result of the decay of high-energy compounds and depletion of energy reserves in dogs with pancreatitis.

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### ЛІПІДНИЙ ПРОФІЛЬ КРОВІ ЯК ДІАГНОСТИЧНИЙ МАРКЕР ГОСТРОГО ПАНКРЕАТИТУ СОБАК

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**Анотація.** Гострий панкреатит належить до поширених незаразних патологій собак, несвоєчасна діагностика та терапія якого закінчується летально (27–58 % випадків). Одним з найпоширеніших діагностичних тестів гострого панкреатиту є визначення концентрації тригліцеридів та інших фракцій ліпідів крові. Метою досліджень було дослідити зміни вмісту фракцій ліпідів та інших біохімічних показників у собак за гострого панкреатиту. Для експерименту було сформовано дві групи собак по 10 особин в кожній. Контрольна група включала здорових тварин, дослідна – собак із гострим панкреатитом. Встановлено, що у собак з гострим панкреатитом концентрація тригліцеридів зростала на 67 % порівняно зі здоровими особинами. Концентрація холестерину і фосфоліпідів також збільшувалась на 23 і 9 % відповідно. Запальні процеси у підшлунковій залозі призводили до розвитку цитолітичних процесів у її ацинусах, а також гепатоцитах печінки, на що вказувала збільшена активність індикаторних ферментів АлАТ, АсАТ, ЛФ та білірубину. Також у хворих собак порушувалась білоксинтезувальна функція печінки: реєстрували зменшену частку альбумінів на фоні підвищеного загального білка сироватки крові. Гострий панкреатит призводить до зменшення синтезу інсуліну, що проявляється гіперглікемією – у хворих собак рівень глюкози вірогідно вдвічі перевищив показник здорових тварин. За панкреатиту порушення перетравлення та всмоктування поживних речовин корму обумовлювало інтенсивне використання організмом макроергів (зокрема, креатинфосфату) і збільшення креатиніну (у 3,8 рази). Водночас зростав катаболізм білків і накопичувались продукти їх проміжного та кінцевого обміну – аміак і сечовина (на 60 та 57 % відповідно), що є ознакою зменшення фільтраційної здатності нирок. Крім того, за гострого панкреатиту в межах фізіологічної норми концентрація Кальцію зменшувалась, а неорганічного фосфору – збільшувалась

**Ключові слова:** тригліцериди, холестерин, фосфоліпіди, індикаторні ферменти, підшлункова залоза

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